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NEWS	17	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	18	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS	19	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	20	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS	21	FEB 28	TOXCENTER reloaded with enhancements
NEWS	22	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS	23	MAR 01	INSPEC reloaded and enhanced
NEWS	24	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes

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=> s ginkgolide (p) breast
626 GINKGOLIDE
66694 BREAST
L1 3 GINKGOLIDE (P) BREAST

=> d 1-3 ab

L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AB A review. Recent studies conducted with various mol., cellular and whole animal models have revealed that leaf exts. of Ginkgo biloba may have anticancer (chemopreventive) properties that are related to their antioxidant, anti-angiogenic and gene-regulatory actions. The antioxidant and associated anti-lipoperoxidative effects of Ginkgo exts. appear to involve both their flavonoid and terpenoid constituents. The anti-angiogenic activity of the exts. may involve their antioxidant activity and their ability to inhibit both inducible and endothelial forms of nitric oxide synthase. With regard to gene expression, a Ginkgo extract and one of its terpenoid constituents, **ginkgolide** B, inhibited the proliferation of a highly aggressive human **breast** cancer cell line and xenografts of this cell line in nude mice. CDNA microarray analyses have shown that exposure of human **breast** cancer cells to a Ginkgo extract altered the expression of genes that are involved in the regulation of cell proliferation, cell differentiation or apoptosis, and that exposure of human bladder cancer cells to a Ginkgo extract produced an adaptive transcriptional response that augments antioxidant status and inhibits DNA damage. In humans, Ginkgo exts. inhibit the formation of radiation-induced (chromosome-damaging) clastogenic factors and UV light-induced oxidative stress - effects that may also be associated with anticancer activity. Flavonoid and terpenoid constituents of Ginkgo exts. may act in a complementary manner to inhibit several carcinogenesis-related processes, and therefore the total exts. may be required for producing optimal effects.

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated **Ginkgolide** B (GKB), a component of the extract of Ginkgo biloba leaves in a method for decreasing the expression of

peripheral-type benzodiazepine receptor (PBR) in cells of a patient in need thereof. Further, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method for decreasing the proliferation of cancer cells in a patient. More particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method of decreasing cancer cell proliferation in a patient wherein the cancer cell is human **breast** cancer cell. Even more particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in method of decreasing cancer cell proliferation in a patient wherein the cancer cell is of the aggressive and invasive phenotype and expresses high levels of PBR in comparison to non-aggressive cancer cell.

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

AB The peripheral-type benzodiazepine receptor (PBR) expression and localization correlate with human **breast** cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated **ginkgolide** B (GKB) were shown to decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human **breast** cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human **breast** cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human **breast** cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

=> d 1-3

L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:594452 CAPLUS
DN 140:35147
TI Ginkgo biloba extracts and cancer: A research area in its infancy
AU DeFeudis, Francis V.; Papadopoulos, Vassilios; Drieu, Katy
CS Institute for BioScience, Westboro, MA, USA
SO Fundamental & Clinical Pharmacology (2003), 17(4), 405-417
CODEN: FCPHEZ; ISSN: 0767-3981
PB Blackwell Publishing Ltd.
DT Journal; General Review
LA English
RE.CNT 106 THERE ARE 106 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:137040 CAPLUS
DN 134:173024
TI Ginkgo extract for cancer treatment
IN Drieu, Katy; Papadopoulos, Vassilios
PA Societe de Conseils de Recherches et d'Applications Scientifiques, S.A.S., Fr.; Georgetown University
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001012208	A1	20010222	WO 2000-US22174	20000811
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
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CA	2378052	AA	20010222	CA 2000-2378052	20000811
EP	1200108	A1	20020502	EP 2000-955489	20000811
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP	2003507336	T2	20030225	JP 2001-516553	20000811
NO	2002000666	A	20020211	NO 2002-666	20020211
PRAI US	1999-148604P	P	19990812		
	WO 2000-US22174	W	20000811		

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:783981 CAPLUS
DN 134:320580
TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor expression and cell proliferation in human breast cancer cells
AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick, Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau, Jacque-Pierre; Drieu, Katy
CS Division of Hormone Research, Georgetown University Medical Center, Washington, DC, 20007, USA
SO Anticancer Research (2000), 20(5A), 2835-2847
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
RE.CNT 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s ginkgolide (p) benzodiazepine
626 GINKGOLIDE
20234 BENZODIAZEPINE
L2 16 GINKGOLIDE (P) BENZODIAZEPINE

=> d 1-16 ab

L2 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB The anxiolytic-like effects of Ginkgo biloba extract (GBE) and its four terpenoid components (**ginkgolide**-A, **ginkgolide**-B, **ginkgolide**-C, and bilobalide) were assessed using the elevated plus-maze test in mice. Administration of GBE as a single oral dose (0.5 or 1 g/kg, po) caused a state of suppressed motor activity and, thus, shortened the time spent in the open-sided arms. However, when GBE (0.063-1 g/kg, po) was administered daily for 7 days and the plus-maze test was carried out 24 h after the final administration, the time spent in the open-sided arms was prolonged, with the peak anxiolytic-like effect at 0.125 g/kg. A combination of seven-day administration of GBE (0.125 g/kg) and a single dose of diazepam (1 mg/ kg, po, 10 min before testing) enhanced the anxiolytic-like effect. Flumazenil (0.3 mg/kg, i.p., 10 min before testing) blocked the effect of diazepam, but not of GBE. Daily administration of **ginkgolide**-A (1 or 2 mg/kg, po) resulted in an anxiolytic-like effect by the third treatment, with the maximal effect observed after the fifth administration. Neither **ginkgolide**-B, **ginkgolide**-C, nor bilobalide produced any anxiolytic-like effects. At doses higher than 0.5 g/kg, GBE not only inhibited motor activity but also suppressed active avoidance behavior, reduced caffeine-induced stimulation, and enhanced pentobarbital-induced sleep, while **ginkgolide**-A (up to 20 mg/kg) did not exhibit these effects. Diazepam (1 mg/kg) is known to enhance pentobarbital-induced sleep. These results suggest that GBE produces a significant anxiolytic-like effect

following repeated administration and that **ginkgolide**-A is most likely responsible for this effect. There are also indications that although GBE exerts a sedative effect at comparatively higher doses, **ginkgolide**-A has a relatively weak tendency to produce **benzodiazepine**-like side effects.

L2 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Treatment of rats and adrenocortical cells with **ginkgolide** B (GKB), a purified component of Ginkgo biloba leaf exts., reduces the mRNA, protein, and ligand-binding levels of the adrenal peripheral-type **benzodiazepine** receptor (PBR), a mitochondrial cholesterol-binding protein, leading to decreased corticosteroid synthesis. In the Y1 adrenocortical cell line, GKB reduced both PBR levels and cAMP-induced steroid formation. In these cells, GKB, but not various steroids and vitamins, reduced the expression of a reporter gene driven by the DNA sequence -624/-513 relative to the transcription start site of the PBR encoding gene. GKB treatment did not affect the SV40 promoter and increased the cytochrome P 450 17α-hydroxylase gene promoter driven expression of the reporter gene. Electrophoretic mobility shift assays (EMSAs) indicated the presence of a functional transcriptional element bound to the -624/-513 DNA fragment. This GKB-induced inhibition of PBR was mediated by an interaction with a transcription factor that binds to the -636/-616 PBR-promoter region. Deletion or mutation of this sequence eliminated the DNA-protein interaction and the inhibitory effect of GKB on PBR gene transcription. This DNA-binding protein could be detected in nuclear exts. of rat brain, liver, and testis, but not kidney. It is also present in the human adrenal glands. However, the inhibitory effect following GKB treatment could be seen only in the adrenal glands. These results demonstrate that the GKB-activated inhibition of glucocorticoid production is due to a specific transcriptional suppression of the adrenal PBR gene and suggest that GKB might serve as a pharmacol. tool to control excess glucocorticoid formation.

L2 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB A review. Ginkgo biloba leaf extract (EGb 761), which contains many constituents, including flavonoid glycosides and terpenoids (ginkgolides, bilobalide), is used to treat cerebrovascular and peripheral vascular insufficiency, as well as cognitive impairment and other symptoms of dementia. Recent studies have indicated that these therapeutic effects of EGb 761 probably involve modification of the expression of many genes by actions involving several of its active constituents. As examples: EGb 761 and its **ginkgolide** B constituent inhibit the expression of the peripheral **benzodiazepine** receptor in the adrenal cortex and decrease circulating levels of corticosterone in the rat, effects that provide a mechanism for explaining the "antistress" action of the extract Both the flavonoid and terpenoid constituents of EGb 761 decrease the expression of inducible nitric oxide synthase (iNOS), supporting an action of the extract of opposing the deleterious effects of excessive formation of NO. EGb 761 upregulates several genes that encode vital antioxidant enzymes, including heme oxygenase-1 and the regulatory and catalytic subunits of γ-glutamyl-cysteinyl synthetase. Dietary treatment of mice with EGb 761 upregulates the expression of genes encoding neuronal tyrosine/threonine phosphatase 1 and microtubule-associated tau in the cerebral cortex, findings that are of interest since these proteins are associated with the intracellular neurofibrillary tangles found in the brain in Alzheimer's disease. Bilobalide upregulates two mitochondrial-DNA-encoded genes, subunit III of cytochrome c oxidase and subunit ND1 of NADH dehydrogenase, indicating a fundamental mechanism that may underlie EGb 761-induced neuroprotection. Collectively, such results indicate that the therapeutic effects of EGb 761 on cognitive impairment (dementia) may involve its action of altering gene expression.

L2 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB The anxiolytic-like effect of Ginkgo biloba extract (GBE) and its four ginkgo-terpenoids (**ginkgolide**-A, **ginkgolide**-B, **ginkgolide**-C, and bilobalide) were assessed by an improved plus-maze test in mice. The single oral administration of GBE (0.5 and 1 g/kg) shortened the time spent in the open arms with suppression of the motor activity. However, when GBE (0.063-1 g/kg p.o.) was administered

daily for seven days and the plus-maze test was carried out 24 h after the last administration, prolongation of the time spent in the open arms was developed with the peak effect at 0.13 g/kg, showing an anxiolytic-like effect. Diazepam, following the single oral administration at 1 mg/kg, also prolonged the time spent in the open arms. The combination of the seven daily administration of GBE (0.13 g/kg) and single administration of diazepam (1 mg/kg) enhanced the anxiolytic-like effect. Flumazenil (0.3 mg/kg i.p.) blocked the effect of diazepam, but not of GBE. Among the ginkgo-terpenoids, the daily administration of **ginkgolide**-A (1 and 2 mg/kg p.o.) developed the anxiolytic-like effect by the 3rd administration, and the effect achieved to the highest plateau level by the 5th administration. Whereas, the seven daily treatment with **ginkgolide**-B (1 mg/kg), **ginkgolide**-C (1 mg/kg) or bilobalide (1 and 2 mg/kg) caused no anxiolytic-like effect. These results suggest that GBE produces significant anxiolytic-like effect following the repeated administration, and that **ginkgolide**-A is responsible for this effect. However, it is unlike that **benzodiazepine** receptors are involved in the development of the anxiolytic-like effect of GBE and **ginkgolide**-A.

L2 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Identification of the mol. switch controlling glucocorticoid synthesis might facilitate the development of pharmacol. tools to control circulating cortisol levels. The transport of cholesterol from intracellular sources to the inner mitochondrial membrane represents the rate-determining step in the cascade of reactions leading to cortisol synthesis. A key element in this step is the peripheral-type **benzodiazepine** receptor (PBR). Several studies have indicated the beneficial effects of Ginkgo biloba on memory and stress control. Using pharmacol., biochem. and proteomic methods, we demonstrated that the standardized Ginkgo biloba extract EGb 761 and its isolated component **ginkgolide** B (GKB) inhibit PBR ligand binding and protein expression, resulting in decreased serum corticosterone levels. We further demonstrated that EGb 761- and GKB-induced inhibition of PBR protein is preceded by a decrease in mRNA-levels due to transcriptional suppression of PBR gene expression. Further studies indicated that the action of GKB is mediated by a transcription factor binding to the PBR gene promoter, thereby regulating PBR gene expression. These data indicate that EGb 761-induced inhibition of glucocorticoid production is due to specific transcriptional suppression of the adrenal PBR gene by GKB, and suggest that EGb 761 and GKB might serve as pharmacol. tools to control excess glucocorticoid formation.

L2 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB This invention relates to the use of ginkgolides and exts. of Ginkgo biloba for inhibiting the membrane expression of benzodiazepine receptors and for inhibiting the release of glucocorticoids in a patient.

L2 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated **Ginkgolide** B (GKB), a component of the extract of Ginkgo biloba leaves in a method for decreasing the expression of peripheral-type **benzodiazepine** receptor (PBR) in cells of a patient in need thereof. Further, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method for decreasing the proliferation of cancer cells in a patient. More particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method of decreasing cancer cell proliferation in a patient wherein the cancer cell is human breast cancer cell. Even more particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in method of decreasing cancer cell proliferation in a patient wherein the cancer cell is of the aggressive and invasive phenotype and expresses high levels of PBR in comparison to non-aggressive cancer cell.

L2 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB The peripheral-type **benzodiazepine** receptor (PBR) expression and localization correlate with human breast cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated **ginkgolide** B (GKB) were shown to

decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human breast cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human breast cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human breast cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

L2 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB A review with 140 refs. The effects of EGb 761 on the CNS underlie one of its major therapeutic indications; i.e., individuals suffering from deteriorating cerebral mechanisms related to age-associated impairments of memory, attention and other cognitive functions. EGb 761 is currently used as symptomatic treatment for cerebral insufficiency that occurs during normal ageing or which may be due to degenerative dementia, vascular dementia or mixed forms of both, and for neuro-sensory disturbances. Depressive symptoms of patients with Alzheimer's disease (AD) and aged non-Alzheimer patients may also respond to treatment with EGb 761 since this extract has an "anti-stress" effect. Basic and clin. studies, conducted both in vitro and in vivo, support these beneficial neuroprotective effects of EGb 761. EGb 761 has several major actions; it enhances cognition, improves blood rheol. and tissue metabolism, and opposes the detrimental effects of ischemia. Several mechanisms of action are useful in explaining how EGb 761 benefits patients with AD and other age-related, neurodegenerative disorders. In animals, EGb 761 possesses antioxidant and free radical-scavenging activities, it reverses age-related losses in brain α1-adrenergic, 5-HT1A and muscarinic receptors, protects against ischemic neuronal death, preserves the function of the hippocampal mossy fiber system, increases hippocampal high-affinity choline uptake, inhibits the down-regulation of hippocampal glucocorticoid receptors, enhances neuronal plasticity, and counteracts the cognitive deficits that follow stress or traumatic brain injury. Identified chemical constituents of EGb 761 have been associated with certain actions. Both flavonoid and **ginkgolide** constituents are involved in the free radical-scavenging and antioxidant effects of EGb 761 which decrease tissue levels of reactive oxygen species (ROS) and inhibit membrane lipid peroxidn. Regarding EGb 761-induced regulation of cerebral glucose utilization, bilobalide increases the respiratory control ratio of mitochondria by protecting against uncoupling of oxidative phosphorylation, thereby increasing ATP levels, a result that is supported by the finding that bilobalide increases the expression of the mitochondrial DNA-encoded COX III subunit of cytochrome oxidase. With regard to its "anti-stress" effect, EGb 761 acts via its **ginkgolide** constituents to decrease the expression of the peripheral **benzodiazepine** receptor (PBR) of the adrenal cortex.

L2 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB In various steroidogenic cell models, mitochondrial prepns. and submitochondrial fractions, the expression of the mitochondrial 18 kDa peripheral-type **benzodiazepine** receptor (PBR) protein confers the ability to take up and release, upon ligand activation, cholesterol. Thus, cholesterol becomes available to P450scc on the inner mitochondrial membrane. These in vitro studies were validated by in vivo expts. Treatment of rats with **ginkgolide** B (GKB), specifically reduced the ligand binding capacity, protein, and mRNA expression of the adrenocortical PBR and circulating glucocorticoid levels. Treatment with GKB also resulted in inhibition of PBR protein synthesis and corticosterone production by isolated adrenocortical cells in response to ACTH. The ontogeny of both PBR binding capacity and protein directly

paralleled that of ACTH-inducible steroidogenesis in rat adrenal cells and in rats injected with ACTH. In addition, the previously described suppression of luteal progesterone synthesis in the pregnant rat by continuous in vivo administration of a gonadotropin-releasing hormone agonist may be due to decreased luteal PBR ligand binding and mRNA. These results suggest that (i) PBR is an absolute prerequisite for adrenocortical and luteal steroidogenesis, (ii) regulation of adrenal PBR expression may be used as a tool to control circulating glucocorticoid levels and (iii) the stress hypo-responsive period of neonatal rats may result from decreased adrenal cortical PBR expression.

L2 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB The hypersecretion of glucocorticoids during exposure to various stressors may induce or worsen pathol. states in predisposed subjects. Therefore it is of interest to evaluate drugs able to reduce glucocorticoid secretion. It has recently been shown that chronic administration of a Ginkgo biloba extract (EGb 761) inhibits stress-induced corticosterone hypersecretion through a reduction in the number of adrenal peripheral **benzodiazepine** receptors. The present study was designed to analyze the effect of EGb 761 and one of its components, **Ginkgolide** B on the biosynthesis and secretion of CRH and AVP, the hypothalamic neurohormones that regulate the pituitary-adrenal axis. Chronic administration of EGb 761 (50 or 100 mg/kg p.o. daily for 14 days) reduced basal corticosterone secretion and the subsequent increase in CRH and AVP gene expression. Under the same conditions, surgically-induced increase in CRH secretion was attenuated while the activation of CRH gene expression, ACTH and corticosterone secretion following insulin-induced hypoglycemia remained unchanged. Chronic i.p. injection of **Ginkgolide** B reduced basal corticosterone secretion without alteration in the subsequent CRH and AVP increase. However, the stimulation of CRH gene expression by insulin-induced hypoglycemia was attenuated by **Ginkgolide** B. These data confirm that the administration of EGb 761 and **Ginkgolide** B reduces corticosterone secretion. In addition, these substances act also at the hypothalamic level and are able to reduce CRH expression and secretion. However the latter effect appears to be complex and may depend upon both the nature of stress and substance (**Ginkgolide** B or other compds. of EGb 761).

L2 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB It was previously demonstrated that repeated treatment of rats with the standardized extract of Ginkgo biloba leaves, EGb 761, and its bioactive component **ginkgolide** B (GKB), specifically reduces the ligand binding, and protein and mRNA expression of the adrenal mitochondrial peripheral **benzodiazepine** receptor (PBR), a key element in the regulation of cholesterol transport, resulting in decreased circulating corticosterone levels. Adrenocortical cells were isolated from rats treated with EGb 761 or GKB and cultured for 2 and 12 days. The effect of ACTH on normal and metabolically labeled cells was examined Corticosterone levels were measured by RIA, and protein synthesis was analyzed by two-dimensional gel electrophoresis. Ex vivo treatment with EGb 761 and GKB resulted, resp., in 50% and 80% redns. of ACTH-stimulated corticosterone production by adrenocortical cells cultured for 2 days compared with that by cells isolated from saline-treated rats. Two-dimensional gel electrophoresis anal. revealed that in cells from both control and drug-treated animals, ACTH induced the synthesis, at the same level, of a 29-kDa and pI 6.4-6.7 protein identified as the adrenal steroidogenic acute regulatory protein (StAR). In addition, treatment with EGb 761 and GKB specifically altered the synthesis of seven proteins, including inhibition of synthesis of a 17-kDa, identified as PBR. After 12 days in culture, ACTH-stimulated adrenocortical cell steroid synthesis was maintained, and it was identical among the cells isolated from animals treated with GKB or saline. Under the same conditions, the expression of PBR was recovered, whereas no effect of ACTH on the 29-kDa and 6.4-6.7 pI protein (StAR) or other protein synthesis could be seen. A comparative anal. of the effects of GKB and EGb 761 on adrenocortical steroidogenesis and protein synthesis identified, in addition to the 17-kDa PBR, target proteins of 32 kDa (pI 6.7) and 40 kDa (pI 5.7-6.0) as potential mediators of the effect of EGb 761 and GKB on ACTH-stimulated glucocorticoid synthesis. In conclusion, these results (1) validate and extend our previous in vivo findings on the

effect of EGb 761 and GKB on ACTH-stimulated adrenocortical steroidogenesis, (2) demonstrate the specificity and reversibility of EGb 761 and GKB treatment, (3) question the role of the 29-kDa, 6.4-6.7 pI protein (mature StAR) as the sole mediator of ACTH-stimulated steroid production, and (4) demonstrate the obligatory role of PBR in hormone-regulated steroidogenesis.

L2 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Ginkgolides may be used to inhibit the membrane expression of a **benzodiazepine** receptor in a patient and in treatments to combat excess glucocorticoid production A Ginkgo biloba extract, **ginkgolide** A, and **ginkgolide** B were tested for their ability to decrease the number of binding sites for the peripheral **benzodiazepine** receptor ligand PK 11195, which binds to an 18 kDa peripheral **benzodiazepine** receptor protein in adrenal mitochondria; expression of the receptor was reduced by 40, 50, and 73%, resp. Inhibition of glucocorticoid (e.g. corticosterone) production by **ginkgolide** A and **ginkgolide** B is also shown, as well as a corresponding increase in ACTH.

L2 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Glucocorticoid excess has broad pathogenic potential including neurotoxicity, neuroendangerment, and immunosuppression. Glucocorticoid synthesis is regulated by ACTH, which acts by accelerating the transport of the precursor cholesterol to the mitochondria where steroidogenesis begins. Ginkgo biloba is one of the most ancient trees, and exts. from its leaves have been used in traditional medicine. A standardized extract of Ginkgo biloba leaves, termed EGb 761 (EGb), has been shown to have neuroprotective and antistress effects. In vivo treatment of rats with EGb, and its bioactive components **ginkgolide** A and B, specifically reduces the ligand binding capacity, protein, and mRNA expression of the adrenocortical mitochondrial peripheral-type **benzodiazepine** receptor (PBR), a key element in the regulation of cholesterol transport, resulting in decreased corticosteroid synthesis. As expected, the **ginkgolide**-induced decrease in glucocorticoid levels resulted in increased ACTH release, which in turn induced the expression of the steroidogenic acute regulatory protein. Because ginkgolides reduced the adrenal PBR expression and corticosterone synthesis despite the presence of high levels of steroidogenic acute regulatory protein, these data demonstrate that PBR is indispensable for normal adrenal function. In addition, these results suggest that manipulation of PBR expression could control circulating glucocorticoid levels, and that the antistress and neuroprotective effects of EGb are caused by its effect on glucocorticoid biosynthesis.

L2 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Pruritis is treated by administration of a therapeutically effective amount of a platelet-activating factor (I) antagonist. The I antagonist may be e.g. a synthetic I analog, a natural product, or a triazolothienodiazepine. I-induced pruritis was blocked by CV-6209 (synthetic I analog), BN 52021 (ginkgolide B), and WEB 2086 (a triazolothienodiazepine derivative), but not by pyrilamine.

L2 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AB Platelet activating factor (PAF) is ubiquitous in mammals, and may have multiple functions in the central nervous system. Triazolobenzodiazepine compds. are active both at the GABAA receptor and as PAF antagonists. To investigate whether PAF antagonist activity is involved in the actions of triazolobenzodiazepines, the effects of two nonbenzodiazepine PAF antagonists on binding and function at the GABAA receptor were investigated. The **ginkgolide** terpene, BN52021 and the dioxolane-based compound BN 52115 had no effect on **benzodiazepine** binding or chloride channel binding in cortical membrane prepns. However, chloride uptake into cortical synaptoneurosomes was enhanced with 1 uM BN 52021 but not 1 uM BN 52115. The effect of BN 52021 was prevented by 1 uM flumazenil. PAF antagonists appear to augment GABAA receptor function without affecting binding.

=> d 1-16

L2 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:761598 CAPLUS
DN 139:374862
TI An Anxiolytic-Like Effect of Ginkgo biloba Extract and Its Constituent, Ginkgolide-A, in Mice
AU Kuribara, Hisashi; Weintraub, Susan T.; Yoshihama, Tatsumi; Maruyama, Yuji
CS Center for Cooperative Research, Medical Division, Gunma University, Showa, Maebashi, Gunma, 371-8511, Japan
SO Journal of Natural Products (2003), 66(10), 1333-1337
CODEN: JNPRDF; ISSN: 0163-3864
PB American Chemical Society
DT Journal
LA English
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:164618 CAPLUS
DN 139:79074
TI Transcriptional suppression of the adrenal cortical peripheral-type **benzodiazepine** receptor gene and inhibition of steroid synthesis by **ginkgolide** B
AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios
CS Department of Cell Biology, Division of Hormone Research, Georgetown University Medical Center, Washington, DC, 20057, USA
SO Biochemical Pharmacology (2003), 65(5), 717-729
CODEN: BCPCA6; ISSN: 0006-2952
PB Elsevier Science Inc.
DT Journal
LA English
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:122597 CAPLUS
DN 139:62369
TI Effects of Ginkgo biloba extract (EGb 761) on gene expression: Possible relevance to neurological disorders and age-associated cognitive impairment
AU DeFeudis, Francis V.
CS Institute for BioScience, Westboro, MA, 01581, USA
SO Drug Development Research (2002), 57(4), 214-235
CODEN: DDREDK; ISSN: 0272-4391
PB Wiley-Liss, Inc.
DT Journal; General Review
LA English
RE.CNT 187 THERE ARE 187 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:33171 CAPLUS
DN 139:143745
TI The anxiolytic-like effect of Ginkgo biloba extract and the constituents assessed by an improved plus-maze test in mice
AU Kuribara, Hisashi; Maruyama, Yuji; Yoshihama, Tatsumi
CS Center for Cooperative Research, Medical Division, Gunma University, Japan
SO Japanese Pharmacology & Therapeutics (2002), 30(11), 955-962
CODEN: JPTABU
PB Raifu Saiensu Shuppan K.K.
DT Journal
LA Japanese

L2 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:805581 CAPLUS
DN 138:314440
TI Use of **ginkgolide** B and a **ginkgolide**-activated response element to control gene transcription: Example of the

adrenocortical peripheral-type **benzodiazepine** receptor
AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios
CS Institut Henri Beaufour Ipsen, Paris, Fr.
SO Cellular and Molecular Biology (Paris, France, Printed) (2002), 48(6), 633-639
CODEN: CMOBEF; ISSN: 0145-5680
PB CMB Association
DT Journal
LA English
RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:593283 CAPLUS
DN 135:157669
TI Ginkgolides for inhibition of membrane expression of benzodiazepine receptors
IN Drieu, Katy
PA Societe de Conseils de Recherches et d'Applications Scientifiques (SCRAS), Fr.
SO U.S., 5 pp., Cont.-in-part of U.S. Ser. No. 575,902, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6274621	B1	20010814	US 1998-68368	19980723
TW 513305	B	20021211	TW 1996-85113522	19961105
WO 9717068	A1	19970515	WO 1996-EP5005	19961108
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
ZA 9609437	A	19970618	ZA 1996-9437	19961109
US 2002006955	A1	20020117	US 2001-879306	20010612
US 2005281899	A1	20051222	US 2005-130680	20050517
PRAI US 1995-7337P	P	19951109		
US 1995-575902	B2	19951220		
WO 1996-EP5005	W	19961108		
US 1998-68368	A1	19980723		
US 2001-879306	A1	20010612		

RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:137040 CAPLUS
DN 134:173024
TI Ginkgo extract for cancer treatment
IN Drieu, Katy; Papadopoulos, Vassilios
PA Societe de Conseils de Recherches et d'Applications Scientifiques, S.A.S., Fr.; Georgetown University
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001012208	A1	20010222	WO 2000-US22174	20000811
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA	2378052	AA	20010222	CA 2000-2378052	20000811
EP	1200108	A1	20020502	EP 2000-955489	20000811
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP	2003507336	T2	20030225	JP 2001-516553	20000811
NO	2002000666	A	20020211	NO 2002-666	20020211
PRAI US	1999-148604P	P	19990812		
	WO 2000-US22174	W	20000811		

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:783981 CAPLUS
DN 134:320580
TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor expression and cell proliferation in human breast cancer cells
AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick, Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau, Jacque-Pierre; Drieu, Katy
CS Division of Hormone Research, Georgetown University Medical Center, Washington, DC, 20007, USA
SO Anticancer Research (2000), 20(5A), 2835-2847
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
RE.CNT 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:521260 CAPLUS
DN 133:207145
TI Ginkgo biloba extract (EGb 761) and CNS functions: basic studies and clinical applications
AU DeFeudis, F. V.; Drieu, K.
CS Institute for BioScience, Westboro, MA, 01581, USA
SO Current Drug Targets (2000), 1(1), 25-58
CODEN: CDTUAU; ISSN: 1389-4501
PB Bentham Science Publishers Ltd.
DT Journal; General Review
LA English
RE.CNT 166 THERE ARE 166 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1999:57991 CAPLUS
DN 130:247179
TI In vivo studies on the role of the peripheral benzodiazepine receptor (PBR) in steroidogenesis
AU Papadopoulos, V.; Widmaier, E. P.; Amri, H.; Zilz, A.; Li, H.; Culty, M.; Castello, R.; Philip, G. H.; Sridaran, R.; Drieu, K.
CS Departments of Cell Biology & Pharmacology, Georgetown University Medical Center, Washington, DC, USA
SO Endocrine Research (1998), 24(3 & 4), 479-487
CODEN: ENRSE8; ISSN: 0743-5800
PB Marcel Dekker, Inc.
DT Journal
LA English
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:329598 CAPLUS
DN 129:76458
TI Effect of chronic administration of ginkgo biloba extract or ginkgolide on the hypothalamic-pituitary-adrenal axis in the rat

AU Marcilhac, A.; Dakine, N.; Bourhim, N.; Guillaume, V.; Grino, M.; Drieu, K.; Oliver, C.
CS Laboratoire de Neuroendocinologie Experimentale, Institut Jean Roche, INSERM U 297, Faculte de Medecine Ecteur Nord, Marseille, 13916, Fr.
SO Life Sciences (1998), 62(25), 2329-2340
CODEN: LIFSAK; ISSN: 0024-3205
PB Elsevier Science Inc.
DT Journal
LA English
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1997:757955 CAPLUS
DN 128:57588
TI Ex vivo regulation of adrenal cortical cell steroid and protein synthesis, in response to adrenocorticotropic hormone stimulation, by the Ginkgo biloba extract EGb 761 and isolated ginkgolide B
AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios
CS Deps. Cell Biol., Georgetown Univ. Med. Cent., Washington, DC, 20007, USA
SO Endocrinology (1997), 138(12), 5415-5426
CODEN: ENDOAO; ISSN: 0013-7227
PB Endocrine Society
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1997:414183 CAPLUS
DN 127:29095
TI Ginkgolides for inhibition of membrane expression of benzodiazepine receptors and to combat excess glucocorticoid production
IN Drieu, Katy
PA Societe de Conseils de Recherches et d'Applications Scientifiques (SCRAS), Fr.; Cockbain, Julian
SO PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9717068	A1	19970515	WO 1996-EP5005	19961108
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	TW 513305	B	20021211	TW 1996-85113522	19961105
	CA 2235416	AA	19970515	CA 1996-2235416	19961108
	AU 9675717	A1	19970529	AU 1996-75717	19961108
	AU 724416	B2	20000921		
	EP 862428	A1	19980909	EP 1996-938209	19961108
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000505057	T2	20000425	JP 1997-517873	19961108
	NZ 322050	A	20000728	NZ 1996-322050	19961108
	ZA 9609437	A	19970618	ZA 1996-9437	19961109
	US 6274621	B1	20010814	US 1998-68368	19980723
	US 2002006955	A1	20020117	US 2001-879306	20010612
	US 2005281899	A1	20051222	US 2005-130680	20050517
PRAI	US 1995-7337P	P	19951109		
	US 1995-575902	A2	19951220		
	WO 1996-EP5005	W	19961108		
	US 1998-68368	A1	19980723		
	US 2001-879306	A1	20010612		

OS MARPAT 127:29095

L2 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:726043 CAPLUS
DN 126:69966
TI In vivo regulation of peripheral-type benzodiazepine receptor and glucocorticoid synthesis by Ginkgo biloba extract EGb 761 and isolated ginkgolides
AU Amri, Hakima; Ogwuegbu, Stephen O.; Boujrad, Noureddine; Drieu, Katy; Papadopoulos, Vassilios
CS Dep. Cell Biol., Georgetown Univ. Med. Cent., Washington, DC, 20007, USA
SO Endocrinology (1996), 137(12), 5707-5718
CODEN: ENDOAO; ISSN: 0013-7227
PB Endocrine Society
DT Journal
LA English
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1992:120910 CAPLUS
DN 116:120910
TI Use of platelet-activating factor antagonists as anti-pruritic agents
IN Woodward, David Frederick; Williams, Linda Sue
PA Allergan, Inc., USA
SO PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9118608	A1	19911212	WO 1991-US2003	19910325
	W: AU, BB, BG, BR, CA, FI, HU, JP, KP, KR, LK, MC, MG, MW, NO, PL, RO, SD, SU				
	RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG				
	CA 2083611	AA	19911201	CA 1991-2083611	19910325
	AU 9176908	A1	19911231	AU 1991-76908	19910325
	EP 532512	A1	19930324	EP 1991-907987	19910325
	EP 532512	B1	19961211		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 05507467	T2	19931028	JP 1991-507834	19910325
	JP 3142559	B2	20010307		
	AT 146079	E	19961215	AT 1991-907987	19910325
	ES 2095316	T3	19970216	ES 1991-907987	19910325
PRAI	US 1990-530739	A	19900531		
	WO 1991-US2003	A	19910325		

L2 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1992:99051 CAPLUS
DN 116:99051
TI Platelet activating factor antagonists interact with GABAA receptors
AU Miller, Lawrence G.; Bazan, Nicolas G.; Roy, R. Beth; Clostre, Francois; Gaver, Anat; Braquet, Pierre
CS Med. Cent., LSU, New Orleans, LA, 70112, USA
SO Research Communications in Chemical Pathology and Pharmacology (1991), 74(2), 253-6
CODEN: RCOCB8; ISSN: 0034-5164
DT Journal
LA English

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                   IPC reform
 NEWS  8  DEC 23   New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/
                   USPAT2
 NEWS  9  JAN 13   IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
 NEWS 10  JAN 13   New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to
                   INPADOC
 NEWS 11  JAN 17   Pre-1988 INPI data added to MARPAT
 NEWS 12  JAN 17   IPC 8 in the WPI family of databases including WPIFV
 NEWS 13  JAN 30   Saved answer limit increased
 NEWS 14  JAN 31   Monthly current-awareness alert (SDI) frequency
                   added to TULSA
 NEWS 15  FEB 21   STN AnaVist, Version 1.1, lets you share your STN AnaVist
                   visualization results
 NEWS 16  FEB 22   Status of current WO (PCT) information on STN
 NEWS 17  FEB 22   The IPC thesaurus added to additional patent databases on STN
 NEWS 18  FEB 22   Updates in EPFULL; IPC 8 enhancements added
 NEWS 19  FEB 27   New STN AnaVist pricing effective March 1, 2006
 NEWS 20  FEB 28   MEDLINE/LMEDLINE reload improves functionality
 NEWS 21  FEB 28   TOXCENTER reloaded with enhancements
 NEWS 22  FEB 28   REGISTRY/ZREGISTRY enhanced with more experimental spectral
                   property data
 NEWS 23  MAR 01   INSPEC reloaded and enhanced
 NEWS 24  MAR 03   Updates in PATDPA; addition of IPC 8 data without attributes

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                   CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
                   AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
                   V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
                   http://download.cas.org/express/v8.0-Discover/

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=> S GINKGOLIDE (P) MAMMARY
626 GINKGOLIDE
81960 MAMMARY
L1 1 GINKGOLIDE (P) MAMMARY

=>

=> D 1 AB

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
AB The peripheral-type benzodiazepine receptor (PBR) expression and localization correlate with human breast cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated ginkgolide B (GKB) were shown to decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human breast cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human breast cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human breast cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

=> D 1

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:783981 CAPLUS

DN 134:320580
TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor expression and cell proliferation in human breast cancer cells
AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick, Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau, Jacque-Pierre; Drieu, Katy
CS Division of Hormone Research, Georgetown University Medical Center, Washington, DC, 20007, USA
SO Anticancer Research (2000), 20(5A), 2835-2847
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
RE.CNT 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT